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Flap Necrosis After Large Myelomeningocele Repair Using Good Wound Care and Secondary Intention: Case report

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ABSTRACT

Abstract Myelomeningocele (MMC) is the commonest type of neural tube defect; it represents a protrusion of neural tissue from a bony defect. Treatment of myelomeningocele depends on the size of the defect; with small ones, reconstruction with primary closure or local single flaps may be sufficient, while large (more than 5-8 cm) defects require more complicated reconstructive techniques. Flap necrosis is a major complication of large myelomeningocele repair. In our case, we discuss a new proposed method in post-surgical flap necrosis management based on good wound care, wound coverage, and healing by secondary intention.
Keywords: *Disseminated Staphylococcal Infection, Staphylococcus Aureus, Pediatric Age, Acute Osteomyelitis, Deep Vein Thrombosis, Pulmonary Septic Embolisms*

Introduction

Spina bifida includes a wide spectrum of congenital malformations affecting the spine and the spinal cord, classified as spina bifida aperta (visible lesions, the defect is either not covered completely or covered by a membrane, It includes myelomeningocele.), and spina bifida occulta (non-visible lesions, no apparent defect externally, patients are asymptomatic with no complications and usually do not require treatments) (Alruwaili, 2019).

Myelomeningocele is one of the congenital malformations of the central nervous system. Occurs during embryonic development (fourth week of gestation) due to failure of closure of the spinal neural tube, leading to herniation of the spinal cord or meninges through a vertebral defect with a fluid-filled sac protruding at the level of the defect. The neural tube closure begins at the level of the hindbrain and progresses caudally. Incomplete closure caudally leads to Myelomeningocele formation around day 26 of

gestation. Neurological deficits and complications depend on the involved vertebral level (Alruwaili, 2019). Myelomeningocele is one of the most common birth defects of the central nervous system; its incidence in the United States is about 0.2 to 0.4 per 1,000 live births. The incidence of myelomeningocele increases with lower socioeconomic status and older maternal age (Sahni *et al*., 2023).

Myelomeningocele can be diagnosed after birth by physical examination. Most often diagnosis is made in utero, maternal serum alpha-fetoprotein (MSAFP) test is used as screening test as early as 16 weeks of pregnancy. Definitive diagnosis is made with ultrasound between the 18th and 24th week of pregnancy (Meller *et al*., 2021).

Open fetal surgery takes place between the 19th and 27th week of pregnancy. Women who undergo this surgery are delivered by a Cesarean section around 37 weeks of gestation or earlier if there is an indication for labor, to prevent scar tearing. If fetal surgery is not done, surgical repair after birth is done within the first 48 hours of life to decrease the risk of complications. The repair depends on the size of the defects; the site and shape of the defect, associated spinal disorders, and the status of the surrounding tissues are other important factors that can affect the technique of repair. Most of the soft tissue defects associated with myelomeningocele are small and wound closure with simple soft tissue approximation in the midline is enough. Large defects (more than half the width of the child's back) are not amenable to closure by this simple technique, requiring multilayer muscle, cutaneous or fascial flaps, and grafts. Closure consists of spinal cord repair, closure of the arachnoid and dura, and closure of the soft tissue and the skin. Complications of repair include CSF leak, infection, or contraction of soft tissue. Soft tissue closure with multiple anatomic layers, tension-free soft tissue and skin covering, and natural soft tissue contour restoration can minimize these complications. Surgical repair is performed by neurosurgeons and plastic surgeons (Ritonga and Siahaan, 2022).

In this paper, we discuss a case of large myelomeningocele that was repaired and covered with a skin flap, performed by a neurosurgeon, and complicated with necrosis of the flap. Good care of the skin with antibiotics and skin vitalizers alone was successful in restoring the viability of the skin flap within one and a half months.

Case Presentation

A 3-hour-old female patient presented to our hospital as a case of myelomeningocele and hydrocephalus, the patient was born in a governmental hospital and referred to our hospital for emergent NICU and neurosurgery evaluation and management.

The patient was delivered at 37 +5 by cesarean section for a 36-year-old mother. Apgar score was 8/9 in 1st min and 5th min. her growth parameters at birth were as following (weight = 3500 gm, length = 52 cm, head circumference = 41 cm). The patient's family lives in a rural area and has a very low socioeconomic status. She was lately prenatally diagnosed with myelomeningocele by detailed ultrasound which was done at 27 +6 weeks, it showed spina bifida, - lumbar open, ventriculomegaly (13-15 mm), lemon sign, and banana sign.

The patient arrived at our hospital in respiratory distress (tachypneic and has subcostal retractions) and was put on O2. Other vital signs were stable, Table 1 showing the patient test results on the admission. She was admitted to the NICU, and on examination, it was clear that the baby had an enlarged head, and a large midline defect in the lumbar area from D9 to the sacrum measuring about 12*8 cm (Fig. 1). She was paraplegic, and had bowel and bladder dysfunction, and no anal tone, which indicates a neurological deficit above L3 level. She also had a foot deformity.

Table 1: Pateint's lab at the day of admission, "her first day of life"

Test	Result
WBC count	15.4 * 10^9 /L
Hb	20.9 g/dL
Platelets	207 * 10^9 /L
ESR	0 mm/h
aPTT	31.8 sec
PT	15.5 sec
INR	1.3
pH	7.28
PCO_2	50.9 mmHg
PO_2	75 mmHg
HCO_3-	241 mmol/L
Anion gap	-34 mEq/L
$Ca+^2$	8.6 mg/dl

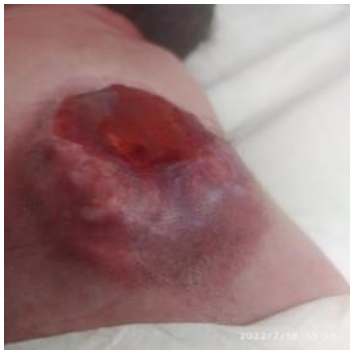

Figure 1: Picture of the patient's MMC and the associated skin defect that was taken before the surgery.

A whole spine CT was done on the admission and showed multi-level defect over nine vertebrae without arch from D9 to the sacrum. Also, a sac-like structure seen connected to the lumbar level appears to be close to the surface, suggestive of meningocele without covering skin (Fig. 2). The brain CT was

performed and showed severe hydrocephalus involving both lateral ventricles. By that, the diagnosis of Arnold Chiari malformation was confirmed. Renal ultrasound showed mild hydronephrosis.

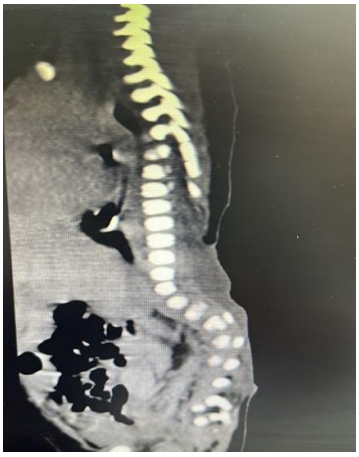

Figure 2: This whole spine CT was done before the surgery, it shows multi-level defect over nine vertebrae without arch from D9 to the sacrum, with a sac-like structure seen connected to the lumbar level appears to be close to the surface, suggestive of myelomeningocele with overlying skin defect.

The patient underwent a classical myelomeningocele repair surgery on the 2nd day of life, the arachnoid and dura matters were closed, the skin defect was repaired by the neurosurgeon only because the plastic surgeon was not available, a large Z skin flap was used with release of all the skin in the lumbar and dorsal area with arriving the skin of the chest and abdomen (Fig. 3), but the tension in the skin couldn't be relieved more than that because the skin defect was large. The baby remained post-operatively in the NICU and was managed with daily dressing and regular neurosurgeon follow-up. On the 29th day, a VP shunt was placed to relieve hydrocephalus.

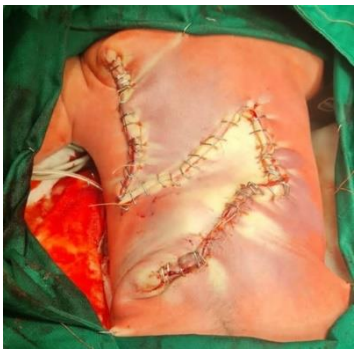

Figure 3: This image is taken for the patient immediately after the surgery, it shows the skin repair with a large Z-flap.

Since the admission, the baby was given a 10-day course of antibiotics (ampicillin and amikacin), on the 8th day, claforan was added as CSF analysis showed high WBC count and the patient managed as a case of meningitis, and antibiotics continued for other 10 days. Then on the 19th day, the baby's condition

worsened, and septic workup was done, the patient had a positive wound swab for CRE acinetobacter, and positive urine culture for Klebsiella, according to that, she was given a 14-day course of colistin, flucan, and flagyl, then the patient continued colistin and cloxacillin for other 8-day. On the 44th day, the baby developed hypoactivity and was given a 14-day course of ceftazidime and vancomycin, but blood and urine cultures were negative.

During the management of the patient, the skin flap became necrotic 7 days after the surgery (Fig. 4), according to her condition; the large wound, the large necrotic flap, and her increased risk of getting a long-lasting meningitis, it was risky and hard to debride the necrotic skin and use another flap which had had a high chance of failing. The decision was to keep the skin and care for it with twice-daily dressing with hyperoil gel and gentamycin cream, so the skin can heal by secondary intention. The patient continued to be managed in the NICU until two months of age (61 days) and then discharged on hyperoil gel and gentamycin cream twice daily. Despite the patient was not followed as should because the patient was from a rural area and was not attending all follow-up visits, the results were satisfying (Fig. 5).

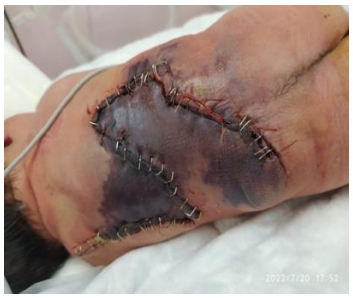

Figure 4: This figure shows the patient's skin flap that became necrotic 7 days after the surgery.

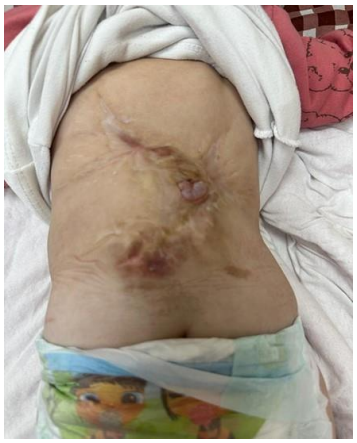

Figure 5: This figure shows the patient's skin flap two months after the surgery, it shows the improvment of the necrotic skin flap with the addressed management.

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Discussion

Myelomeningocele (MMC) is a congenital abnormality of the CNS and represents the most common form of neural tube defect (Farmer *et al*., 2018; Adzick, 2010). It is characterized by the protrusion of the spinal cord and its meninges through open vertebral arches, leading to varying degrees of neurological disability. MMC results from a failure of either neural tube or mesenchymal closure in the caudal neuropore during the fourth week of gestation, resulting in the exposure of the developing neural tissue to the uterine environment (Patel *et al*., 2012). Without adequate protective tissue coverage, these neural tissues are vulnerable to destruction either by trauma or by the amniotic fluid itself, which may leave the infants with lifelong paralysis, bowel and bladder dysfunction, and, various degrees of cognitive disabilities (Farmer *et al*., 2018; Patel *et al*., 2012). Almost all children with MMC develop Chiari II malformation and hydrocephalus (Meller *et al*., 2021).

Myelomeningocele is classified functionally into four subtypes using the Myelomeningocele Functional Classification (MMFC) (Adzick, 2010). MMFC1 may or may not retain their iliopsoas function, but they lack their quadriceps function. These patients need to use a reciprocating gait orthosis (RGO) or a hip-knee-ankle-foot orthosis (AFO) to achieve some ambulation and may need a walker for external support. MMFC2 patients lack gluteus medius function but retain quadriceps and medial hamstring function. Patients need to use walkers and AFOs before the age of four and crutches and AFOs thereafter. MMFC3 patients retain their gluteus medius and quadriceps functions but lack triceps surae function. Most of these patients use AFOs alone without any external support to walk independently. MMFC4 patients preserve the entire lower limb musculature's function and don’t need any assistive devices (Dias *et al*., 2021).

The diagnosis of MMC is done by direct spinal assessment and visualizing the bone defect and the sac protrusion as part of the routine, detailed ultrasound around weeks 18–24. When fetal back visualization is difficult, indirect second-trimester signs of spina bifida should be assessed. These include small biparietal diameter, head circumference, ventriculomegaly, flattened or concave frontal bones ("lemon sign"), obliteration of the cisterna magna, and some cerebellar abnormalities, including a small cerebellum or an anterior concave shape ("banana sign") or the absence of cerebellum in the posterior fossa. The biochemistry panel, including maternal blood alpha-fetoprotein levels, is practically not used anymore. Some new reports suggest the possibility of MMC diagnosis as early as during the 11–14-week period using ultrasound (Meller *et al*., 2021).

Treatment of myelomeningocele can be achieved through prenatal and postnatal surgery to preserve the existing function, cover the exposed spinal cord, eliminate cerebrospinal fluid (CSF) leakage, and

prevent infection. Postnatal surgical treatment depends on the size of the defect. Small defects are closed by skin suturing of the lateral sides in the midline following the management of the neural placode (Ritonga and Siahaan, 2022).

Large defects, defined as being more than half the width of the child's back, need skin or myocutaneous flaps and skin grafts for their closure. Surgical closure of the MMC combined with the treatment of hydrocephalus that is suspected due to the Chiari II malformation. Patients undergo ventricular shunt (VP) surgery at the same time as surgical closure of the MMC defect. Postnatal treatment can be complicated by wound infection, CSF leakage, meningitis, and flap necrosis, which require debridement and/or the use of a new flap (Meller *et al*., 2021; Ritonga and Siahaan, 2022).

Prenatal surgical intervention replicates postnatal surgery in its procedure and is preferred between 19 and 25 weeks of gestation to eliminate the risk of interventional manipulation and preterm labor. Prenatal intervention is expected to decrease neurological deficits in newborn infants by decreasing the likelihood of intrauterine damage to the exposed neural tissues (Meller *et al*., 2021; Ritonga and Siahaan, 2022).

In our case, the patient had a large lumbosacral midline defect measuring about 8*12 cm with a skin defect of about 5*4 cm. There are many different types of flaps mentioned in the literature, and every surgeon has his distinct flap and surgery technic. Our neurosurgeon used to repair these types of MMC defects using Z skin flaps and this was done to our patient. The obtained flap was under tension from day one of surgery. After that, follow-up of the wound with good wound care using skin vetilizers as hyper oil gel for a long time was started. After the first week of surgery. The patient's flap was complicated by flap necrosis, as opposed to what is usually done, the surgeon left the wound without debridement or using another flap. Good wound care with antibiotics and hyperoil gel, combined with wound coverage and good follow-up, were enough to induce wound viability, leaving the wound to heal by secondary intervention, limiting the need for another surgical intervention, and, decreasing the risk of meningitis.

Our suggestion for similar defects, it is better not to manage these complicated surgical wounds with more surgical flaps due to the risk of infection and meningitis. Good Wound care with good antibiotics can achieve wound viability and healing by secondary intention.

Conclusion

Large-defect necrosis is a major complication of myelomeningocele repair surgery. Usually, it is treated by surgical debridement and using new flaps. Good wound care and wound coverage provide a new

proposed method of treating flap necrosis, limiting the need for another major surgery and minimizing the risk of meningitis.

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Conflict of Interest: None declared

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References

Adzick NS. Fetal myelomeningocele: natural history, pathophysiology, and in-utero intervention. *Semin Fetal Neonatal Med* 2010; 15: 9-14.

Alruwaili AA. "Myelomeningocele" (2019).

Dias LS, Swaroop VT, de Angeli LRA, Larson JE, Rojas AM, Karakostas T. Myelomeningocele: a new functional classification. *J Child Orthop* 2021; 15: 1-5

Farmer DL, Thom EA, Brock JW 3rd, Burrows PK, Johnson MP, Howell LJ, Farrell JA, Gupta N, Adzick NS; Management of Myelomeningocele Study Investigators. The Management of Myelomeningocele Study: full cohort 30-month pediatric outcomes. *Am J Obstet Gynecol* 2018; 218: 256.e1-256.e13.

Meller C, Covini D, Aiello H, Izbizky G, Portillo Medina S, Otaño L. Update on prenatal diagnosis and fetal surgery for myelomeningocele. *Arch Argent Pediatr* 2021; 119: e215-e228

Patel KB, Taghinia AH, Proctor MR, Warf BC, Greene AK. Extradural myelomeningocele reconstruction using local turnover fascial flaps and midline linear skin closure. *J Plast Reconstr Aesthet Surg* 2012; 65: 1569-1572.

Ritonga DH, Siahaan AM. Comparison of Prenatal and Postnatal Management in Myelomeningocele: Literature Review. *International Medical Journal* 2022; 29: 233-236.

Sahni M, Alsaleem M, Ohri A. Meningomyelocele. 2023 Sep 4. In: StatPearls [Internet]. Treasure Island (FL): *StatPearls Publishing*; 2023.